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NGR-50-002-0

COMPUTER SIMULATION STUDIES OF THE VENOUS CIRCULATION

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Abstract

An analog computer model of the human cardiovascular system has been set up in which detailed attention is given to the representation of pressure-flow events in the veins, including effects of gravity, collapse, breathing, and venous valves. This model, with its only control loop including one for heart rate control, was checked against human venous pressure waveforms and also response of the human to tilt-table experiments as reported in the literature. These comparisons indicate that the model should be valid for study of postulated venous tone control characteristics, and should be useful in the study of the mechanisms of venous return and circulatory system response during unusual G-force conditions.

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I. INTRODUCTION

The pressure and flow of blood in systmeic veins is of interest to research workers and clinicians concerned with such diverse problems as the changes in venous return which accompany heart failure, and the difficulties encountered by astronauts during flights involving alternate periods of high-g and zero-g conditions [1]. Because of the complexity of the venous system, the mechanisms postulated to explain its action may best be studied with the aid of computer simulations.

The approach adopted in the venous modeling studies made at Wisconsin and at the NASA-Ames Laboratories has the following features:

(a) A pulsatile model is developed for the entire fluid-mechanical cardiovascular system, with most of the detailed emphasis being given to the systemic venous portion of this system.

(b) The model is based on a lumped-constant segmental approximation to the distributed system being modeled, following an approach used by others [2, 3,4] and by these authors in arterial modeling [5].

(c) The venous system portion of the model includes, in addition to the effects of resistance, inertance and compliance, the effects

of collapse, venous valves, gravitational forces, and changes in transmural pressure due to respiration and muscle contractions.

Note that the important venomotor tone effects have not been mentioned as yet. The control loops which result in control of venous volume can be added to this model, and indeed, the use of the model to study such control mechanisms is viewed as one of its principal future applications.

II. METHODS

The basic fluid-mechanical model used in this study is shown in circuit form in Fig. 1. This model consists of four principal parts.

1. The heart, which is represented by time-varying compliances and near-ideal valves. This part of the model does contain one element -- atrial pumping -- which is not usually included in such simple heart simulations, but which is included here because of its importance in systemic venous flow.
2. The pulmonary system, which is rather simply represented, with inertial effects being neglected. Parameter values are based on earlier studies [6, 7].
3. The systemic arterial system, which was also designed as a simplified version of an earlier model [5]. Pulse shapes (e.g. the dicrotic notch) are not particularly realistic in this model, but it was felt that this was not important in the venous studies.
4. The venous systemic circuit, which was rather detailed, including five parallel paths (head and arms, coronaries, upper abdomen, lower abdomen and legs), and the various special venous features such as collapse, etc., mentioned in the introduction. These are

features which are not needed in the arterial system which to date has received most of the computer modeling effort.

It should be explained at this point that the analog computer equipment available to the authors as this paper was written was limited to 192 operational amplifiers, and the detail with which the parts of the system were represented was a kind of compromise based on what was felt to be most important to the objectives of the study.

Various important effects in veins (such as collapse, valve action and gravitational effects) which are not important in arteries had to be included in the model. These will be discussed separately.

(a) Venous Collapse

Venous collapse will be assumed to occur whenever the volume* q of a segment (of length L) becomes less than the unstressed volume Q. The veins will be assumed to be circular in cross-section, with radius r=R at zero static transmural pressure, i.e.,

$$Q = \pi R^2 L$$

For $q \geq Q$ the veins will be assumed to have a constant compliance, as is often assumed for arteries, using values obtained from the literature [8, 9] For $q < Q$ the compliance will be assumed to be 20 times greater, or the slope of the pressure-volume curve will be 20 times less (see Fig. 2). This small value of slope is chosen rather than zero slope, to allow for some wall stiffness and effects of tethering.

*Quantities designated by lowercase letters are variables, i.e. q=q(t), and those by uppercase letters are constants. A glossary of symbols is given in App. I of this paper.

In order to determine the dynamic pressure-flow relationships the effects corresponding to inertance and resistance to flow in the collapsed vein must be determined. In calculating these effects the assumption that during collapse the cross-section of the vein is elliptical with unchanging circumference will be used. The pressure-velocity relationship used in the veins is based on a simple form of the difference-differential equations derived [10] from the Navier-Stokes equations for incompressible fluid flow in cylindrical tubes. This gives, for the relationship between pressure drop Δp and axial velocity w in a segment of a tube of constant radius R and length L, the equation

$$\Delta p = -\rho L \frac{dw}{dt} - \frac{9\mu L}{2R^2} w \qquad (1)$$

where ρ is the density and μ the viscosity of the fluid, and flow has velocity w for $0<r<2R/3$ and is zero for $r>2R/3$ as shown in Fig. 3(a).

The steady-state average velocity in such a tube may be obtained from (1), or a slightly different (and more correct) value from Poiseuille's law,

$$\bar{w}_c = \frac{R^2}{8\mu} \frac{\Delta p}{L} \qquad (2)$$

This may be compared with the value for an elliptical tube of semi-minor and major axes x and y given by Berker [11],

$$\bar{w}_e = \frac{x^2 y^2}{4\mu\left(x^2+y^2\right)} \frac{\Delta p}{L} \qquad (3)$$

Note that R^2 is simply replaced by $2x^2y^2/\left(x^2+y^2\right)$ in going from (2) to (3). Thus it may be argued that, for the collapsing vein, the coefficient of w in (1) should be modified in the same way, yielding

$$\Delta p = - \rho L \frac{dw}{dt} - \frac{9\mu L(x^2+y^2)}{4x^2y^2} w$$

The elliptical cross-section (which will be used to represent the vein during collapse), by analogy with the result given by the difference techniques which give Eq. 1, will be considered to have flow within an inner ellipse of semi-axes 2x/3 and 2y/3 as shown in Fig. 3(b), if q<Q. For q=Q, x=y=R, and Figs. 3(a) and 3(b) will be identical.

The circumference of the elliptical cross-section during collapse is assumed constant and equal to the circumference of the unstressed volume. An approximation to the circumference of an ellipse is $2\pi\sqrt{(x^2+y^2)}/2$. Using this as a constant value equal to $2\pi R$, and using $q=\pi xyL$, the volume of an elliptical segment, in Eq. (4) yields

$$\Delta p = - \rho L \frac{dw}{dt} - \frac{9\mu\pi^2 L^3 R^2}{2q^2} w \qquad (5)$$

for $q \leq Q$.

But we are concerned with flow rather than velocity, and this is given by

$$f = \left(\frac{4q}{9L}\right) w \qquad (6)$$

for q's greater or less than Q. Substituting (6) in (5) gives

$$\Delta p = - \frac{9\rho L^2}{4} \frac{d}{dt}\left(\frac{f}{q}\right) - \frac{81\mu\pi^2 L^4 R^2}{8q^3} f \qquad (7)$$

or

$$f = q\int_0^t \left[- \frac{4\Delta p}{9\rho L^2} - \frac{9\mu\pi^2 L^2 R^2}{2\rho q^3} f \right] dt \qquad (8)$$

for $q \leq Q$. For $q \geq Q$ the cross-section is circular, and Eq. (8) becomes

$$f = q \int_0^t \left[- \frac{4\Delta p}{9\rho L^2} - \frac{9\mu\pi^2 L^2 R^2}{2\rho Q q^2} f \right] dt \qquad (9)$$

which can be derived by using r in place of R and $q=\pi r^2 L$ in (1) and substituting (6) in (1).

These equations show the nonlinear relationship between flow and pressure as volume q in the segment changes during collapse. The inclusion of such nonlinearities in the venous inertances and resistances is indicated in Fig. 1 by the line drawn through the ordinary symbol for these quantities. Collapsible segments considered include the jugular vein, superior vena cava, thoracic vena cava, abdominal vena cava and femoral vein.

(b) Gravity

Appreciable blood volume shifts and changes in pressure can occur even under normal earth gravity as the human changes position, and severe effects can occur with varying acceleration. Thus it is essential that the pressures due to gravity be included in the model. In order not to produce a net pressure around the circulatory loop, these effects must be included in the arteries as well as in the veins, although resultant changes show up much more clearly in the latter.

The value of the pressure generator, p_g, which must be included in series with a segment is the hydrostatic pressure difference over the length of that segment and is given by

$$p_g = nG\rho L \sin\theta \qquad (10)$$

where G is the acceleration due to earth gravity, n is the total of G's of acceleration, ρ is blood density, L is segment length and θ is the angle between the axis of the segment and a perpendicular to the net direction of force.

Because most arteries and veins run side by side it was convenient, in simulating the total circulation, to choose segments of veins and arteries of equal length and to assign equal pressure generators to each.

(c) External Pressure Effects

Intrathoracic and abdominal pressure changes due to respiration, as well as pressure changes due to contraction of muscles in the legs and elsewhere may have important effects on venous flow. All such effects may be represented in the same way in the model, based on the principle that the pressure in a vein or artery with respect to atmospheric pressure is the sum of the transmural pressure and the external pressure. Such external pressures may therefore be included in series with the compliances in the lumped-circuit model.

In the model shown in Fig. 1 intrathoracic pressure sources P_{ITh} are included in series with all of the compliances of vessels in the thoracic cavity. Although not shown in Fig. 1, an intra-abdominal pressure source is included in the same manner. Both pressure sources are a result of respiratory action. A graph of the time course of P_{ITh} as used in the model is shown in Fig. 4. The intra-abdominal pressure source is equal to $-P_{ITh}/2$.

Muscle pumping, which is most important in the legs, is included in the model through the action of a pressure generator P_m in series with the compliances of one venous and one arterial section, as shown in Fig. 1. Because muscle pumping effects are imperfectly known, P_m was varied in the model by hand motion of a potentiometer.

(d) Venous Valves

The effect of venous valves is to inhibit flow away from the heart in veins. Although some valves in larger veins in the extremities are quite competent, the paralleling of paths in the venous system in this model is

such that valves must be simulated as being somewhat less than perfectly competent. This is achieved by use of configurations using two diodes and two resistors as shown in the leg veins in the model of Fig. 1.

(e) Venous Tone Changes

Changes in venomotor tone were included in the model rather simply by varying the unstressed volume(which is equivalent to shifting the pressure-volume curve of Fig. 2 right or left) of the two leg venous segments LVE and LSV based on experiments by Alexander (8). This appears to be the most important part of the venous tone mechanism although there are compliance changes as well, which were not included in the model. A more realistic venous tone control would include abdominal veins and veins of some sections representing skin and muscle blood volume with arterial pressure as the controlling factor. However, present equipment limitations made necessary only manual control of two leg vein segments.

III. RESULTS

The differential and algebraic equations describing the pressures and flows in Fig. 1 were determined and then programmed on an analog computer after choosing appropriate scale factors for the variables. Table 1 shows the values for the resistances, compliances and inertances of Fig. 1. For the collapsible venous segments, resistances and inertances are given for zero transmural pressure, and compliances are given for positive transmural pressure. Time-variable resistances at the entrance of the superior and inferior vena cava into the right atrium were included in the model [12]. Heart rate control was included in the model following the work of Katona [13] and of Dick [14]. Heart rate control was the only feedback control loop included; venomotor tone was only manually controllable.

Table 1

Resistances in $\frac{dynes/cm^2}{ml/sec}$				Compliances in $\frac{ml}{dynes/cm^2} \times 10^6$	
R_1	500	R_{19}	600	C_1	4,000
R_2	88	R_{20}	3,000	C_2	1,200
R_3	535	R_{21}	8,000	C_3	1,700
R_{RA}	10-30	R_{22}	60	C_4	5,300
R_5	15	R_{23}	40	C_5	2,360
R_6	15	R_{24}	25	C_6	2,970
R_7	20	R_{25}	46,000	C_7	225
R_8	120	R_{26}	400	C_8	150
R_9	20	R_{27}	15	C_9	75
R_{10}	15	R_{28}	7,000	C_{10}	75
R_{11}	15	R_{29}	500	C_{11}	1,130
R_{12}	40,000	R_{30}	5,100	C_{12}	1,890
R_{13}	66	R_{31}	300	C_{13}	4,540
R_{14}	40	R_{32}	700	C_{14}	7,200
R_{15}	40	R_{33}	15	C_{15}	187
R_{16}	9,000	R_{34}	20	C_{16}	70
R_{17}	600	R_{35}	30	C_{17}	150
R_{18}	9,000	R_{36}	6,400	C_{18}	150

for $q = Q$

abbr.	collapsible segment	length cm	radius cm	resistance $\frac{dynes/cm^2}{ml/sec}$	inertance $\frac{dynes/cm^2}{ml/sec^2}$	for $q > Q$ compliance $\frac{ml}{dynes/cm^2} \times 10^6$
JV	Jugular Vein	18.	.7	5.73	.789	340
SVC	Superior Vena Cava	1.5	1.0	.115	.032	56
TVC	Thoracic Vena Cava	10.	1.55	.63	.089	900
AVC	Abdominal Vena Cava	16.	1.45	.83	.164	1500
FEV	Femoral Vein	48.	.6	28.0	2.82	750

Pulsatile ventricular volumes and pressures together with two pressures in the systemic veins are shown in Fig. 5. Effects of breathing are included in Fig. 5(b), which shows the considerable increase in ventricular pressures and cardiac output during respiration. Superior and thoracic vena cava pressure waveforms are similar to normally observed waveforms [15, 16] except that in the model the pressure descent following atrial contraction is not as great as is normally observed in the human. Right atrial pressure in the model is essentially equal to the superior vena cava pressure.

Figures 6 and 7 show the response of the model to a simulated tilt-up from 0° to a 90° head-up position, corresponding to a linear increase in acceleration from zero-G to 1G, in about 3.5 seconds (For location of the recorded variables in the model, refer to Fig. 1). The recovery time after tilting back to a horizontal position is much shorter than the response to tilt-up. This response would be expected because of the large stored volume in the systemic veins available almost immediately to the heart when the tilt is reduced back to zero. When the tilt-up is executed recovery volumes must pass through the large peripheral systemic resistance causing a longer time constant. The primary cause of the decrease of all arterial pressures when G=1 in Fig. 6 is the reduction of cardiac output due to the shift of blood into the lower parts of the body. Note in Fig. 7 the presence of collapse when G=1 in the jugular vein as indicated by a negative pressure. The superior vena cava at this time was also collapsed. An approximate idea of cardiac output can be obtained from Figs. 6 and 7 by noting the stroke volume and heart rate. Peak heart rate was set at about 175 per minute with the minimum about 60 per minute.

The pressure changes observed in humans [17] are somewhat less drastic than those observed in the model in Figs. 6 and 7, because of the compensating effects of breathing, venomotor tone and leg muscle contractions on arterial pressures. Fig. 8 shows some of the effects of the inclusion of these influences in the model, all of which tend to shift blood from the lower to the upper vessels increasing the arterial pressures. Breathing, for example, lowers the average pressure in the thoracic vessels, thereby causing a shifting of blood into these vessels and an increase in cardiac output. A venous tone change simulated by a reduction of 40% in both the unstressed volumes, Q, of segments LVE and LSV causes an immediate pressure rise in the corresponding segments forcing blood into the upper vessels and a corresponding rise in cardiac output. Leg muscle contractions, such as occur during walking were simulated in the model by variation of P_m, and also had the effect of increasing pressures in the legs thereby forcing blood upward toward the heart. In the model a magnitude of 90 mm Hg for P_m was applied for a duraction of 2.5 seconds, repeated approximately every 6 seconds.

This model was set up entirely on an analog computer, and exhausted the equipment available (on an AD-256 machine). However, a digital computer linked to the analog is available and will be used to model the additional control loops which must now be incorporated into this model. An all-digital simulation (using CSMP-360) was also tried, but found to be too slow for the study of the long transients encountered in tilt-table experiments.

IV. CONCLUSIONS

An analog computer model of the cardiovascular system has been set up which describes fluid-mechanical events in the venous system in considerable detail. With heart rate control as the only feedback control loop, tests of this model indicate that it duplicates observed pulsatile waveforms quite

well, and gives tilt-table response which agrees reasonably well with what might be expected in the human if vasomotor tone control were absent. It is felt that this model can now be used to compare various control loops postulated for venous tone control and make it possible to add a physiologically accurate loop for such control to the model. With this and other improvements, studies of such things as the effects of unusual G forces on the human, and effects of tilt-table experiments on individuals with circulatory abnormalities may be made by using the model in close collaboration with physiological studies on animals and clinical observations.

V. ACKNOWLEDGEMENTS

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Appendix I

Table of Symbols

L - Length of a segment of vein

Q - Unstressed volume of a segment of vein (circular cross section); $Q = \pi R^2 L$

R - Radius of cross-sectional area of unstressed volume

q - Time variable volume of a segment of vein

w - Time varying axial velocity

$\bar{w}$ - Average velocity in a circular cross section tube

$\bar{w}_e^c$ - Average velocity in an elliptical cross section tube

r - Radius of cross-sectional area for $q > Q$

x,y - Major and minor semi-axes of (elliptical) cross-sectional area for $q < Q$

ρ - Density of blood

μ - Viscosity of blood

Δp - Longitudinal pressure difference

These abbreviations refer to the following segments in the model.

HSV	- Head small veins
JV	- Jugular vein
SVC	- Superior vena cava
UPC, LOC	- Upper and lower carotid artery
RA, LA	- Right and left atrium
RV, LV	- Right and left ventricle
PA, PC, PV	- Pulmonary artery, capillaries and veins
AsA	- Ascending aorta
TVC, ThA	- Thoracic vena cava and aorta
CSMV, CSMA	- Celiac, superior mesenteric vein and artery
PORV	- Portal vein
RENV, RENA	- Renal vein and artery
AVC, AbA	- Abdominal vena cava and aorta
IMV, IMA	- Inferior mesenteric vein and artery
FEV, FA	- Femoral vein and artery
LSV, LSA	- Leg small veins and arteries
LVE, LCAP, LAR	- Leg venules, capillaries and arterioles

Figure Captions

Fig. 1. Fluid-mechanical representation of human closed-loop circulatory system

Fig. 2. Assumed pressure-volume curve for a collapsible vein

Fig. 3. a) Flow in a circular cross-section tube

b) Flow in an elliptical cross-section tube

Fig. 4. Time course of intrathoracic pressure

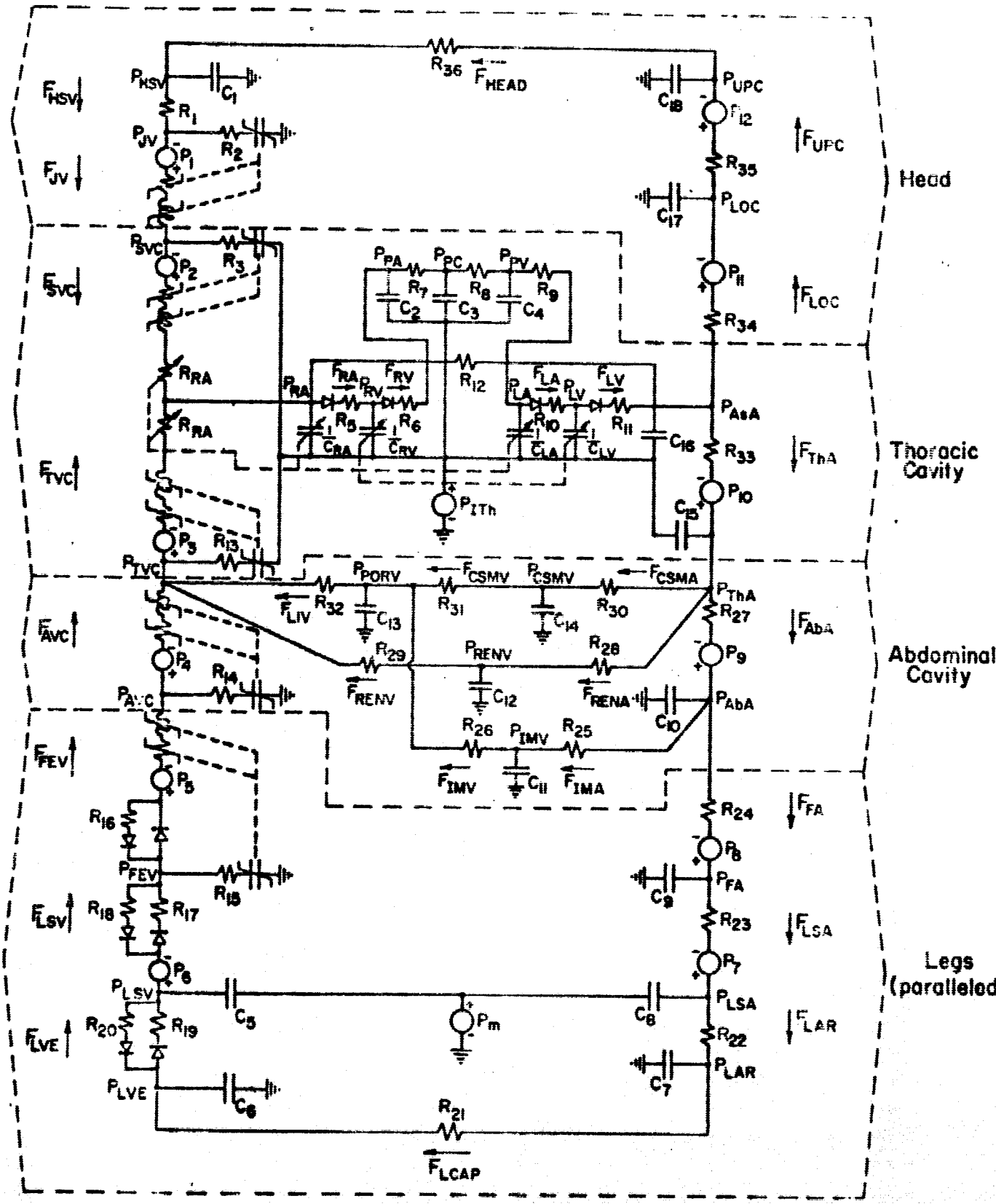
Fig. 5. a) Major variables in right and left heart and systemic veins with zero intrathoracic pressure

b) Major variables in right and left heart and systemic veins with intrathoracic pressure variations due to breathing

Fig. 6. Transients of major arterial pressures with tilt (head up) for the uncontrolled system

Fig. 7. Transients of major venous pressures with tilt (head up) for the uncontrolled system

Fig. 8. Transients of major arterial and venous pressures showing the effects of breathing, reduction of unstressed volume Q corresponding to venous tone changes, and leg muscle contractions in improving cardiac output during tilt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Thoracic Cavity
Abdominal Cavity
Legs (paralleled
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F_{JV}
F_{SVC}
F_{TVC}
F_{AVC}
F_{FEV}
F_{LSV}
F_{LVE}
F_{UPC}
F_{LOC}
F_{ThA}
F_{AbA}
F_{FA}
F_{LSA}
F_{LAR}
F_{HEAD}
F_{LIV}
F_{RENV}
F_{IMV}
F_{CSMV}
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P_{TVC}
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P_{LOC}
P_{AsA}
P_{ThA}
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P_{FA}
P_{LSA}
P_{LAR}
P_{RA}
P_{RV}
P_{LA}
P_{LV}
P_{PA}
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P_{ITh}
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P_{CSMV}
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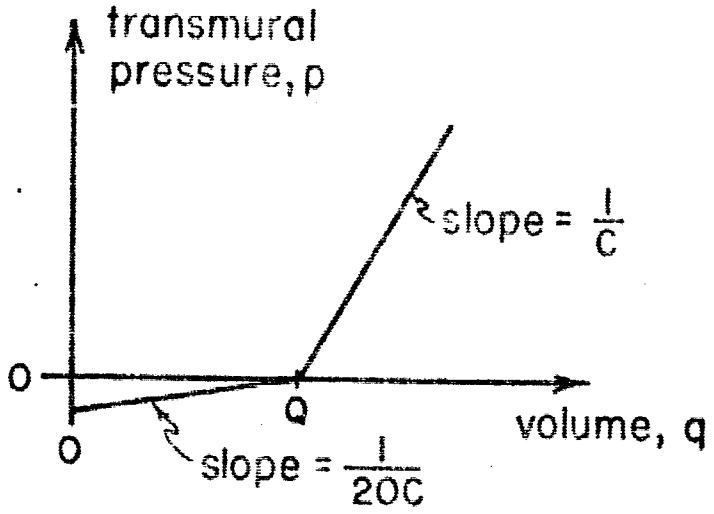
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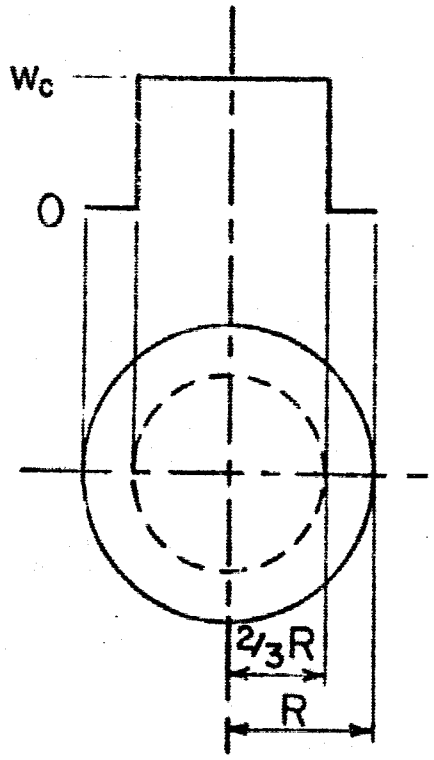
slope = 1/C
0
Q
0
volume, q
slope = 1/2C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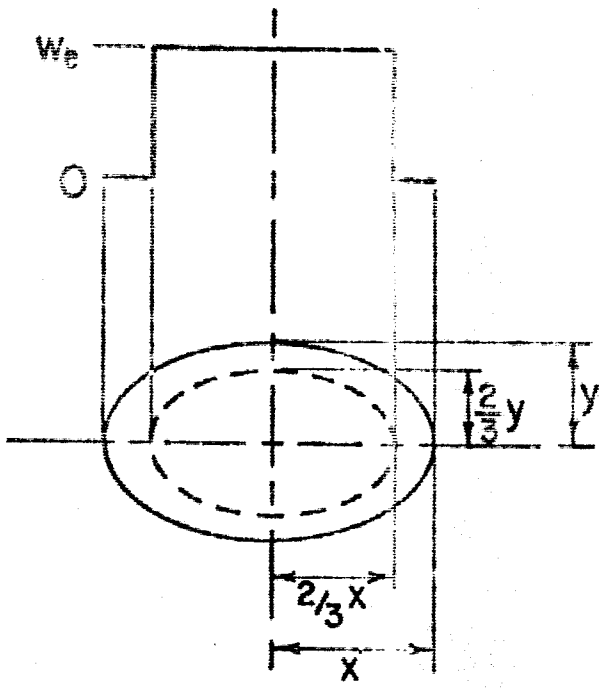
(a)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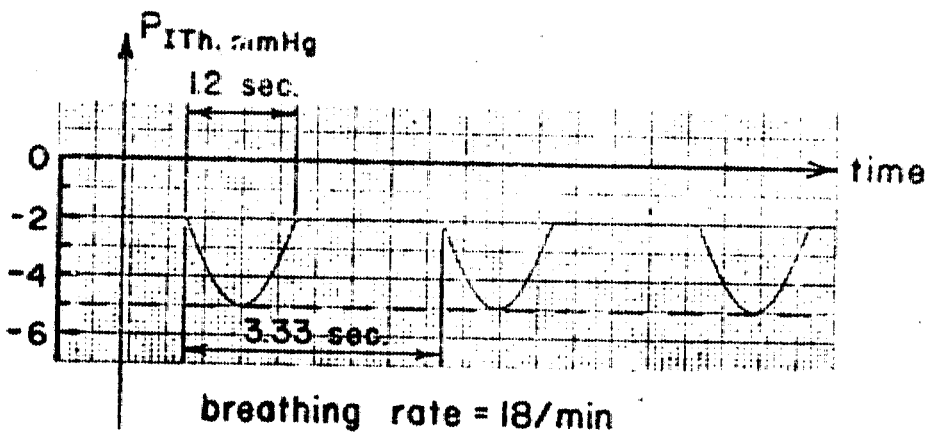
(b)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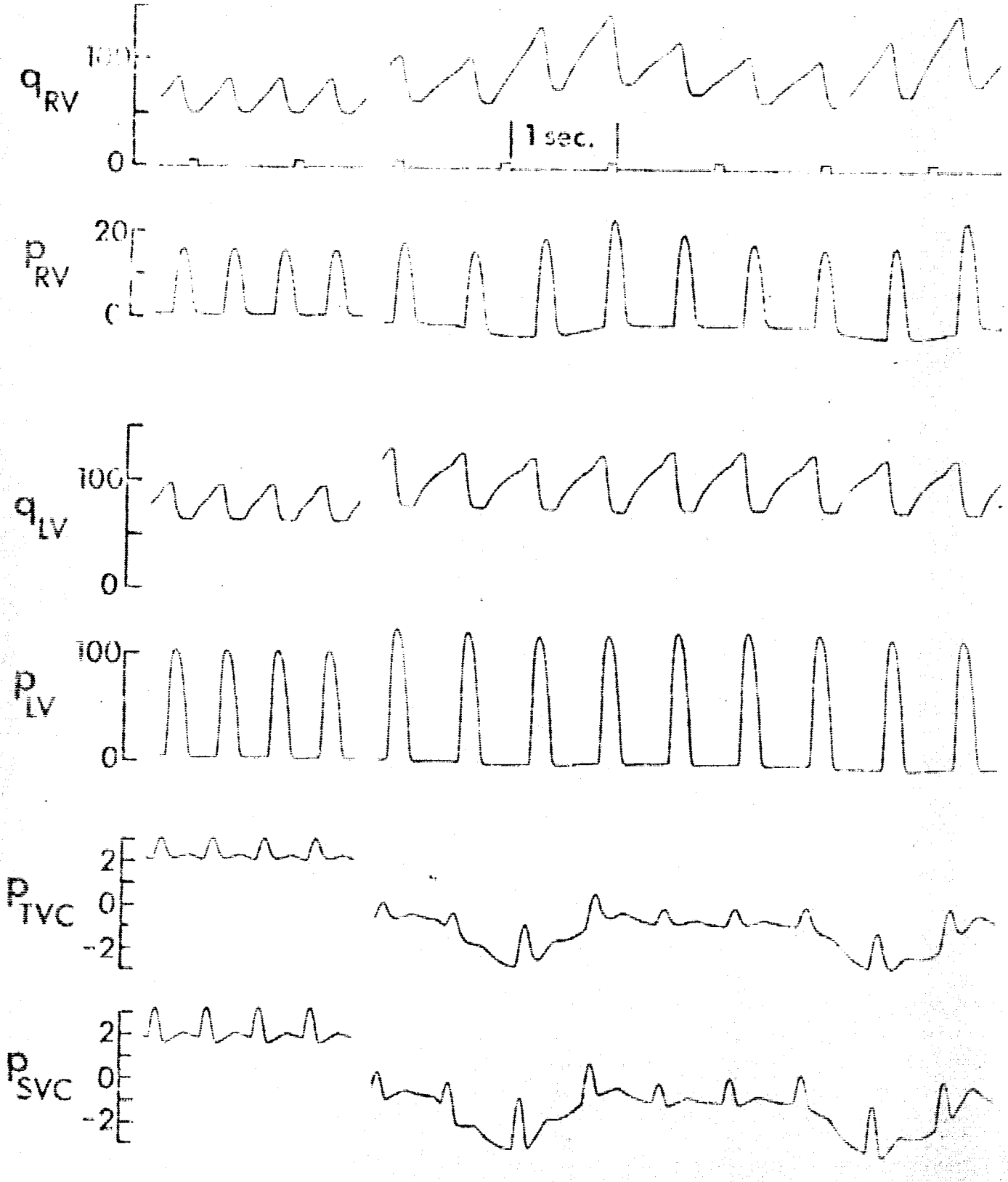
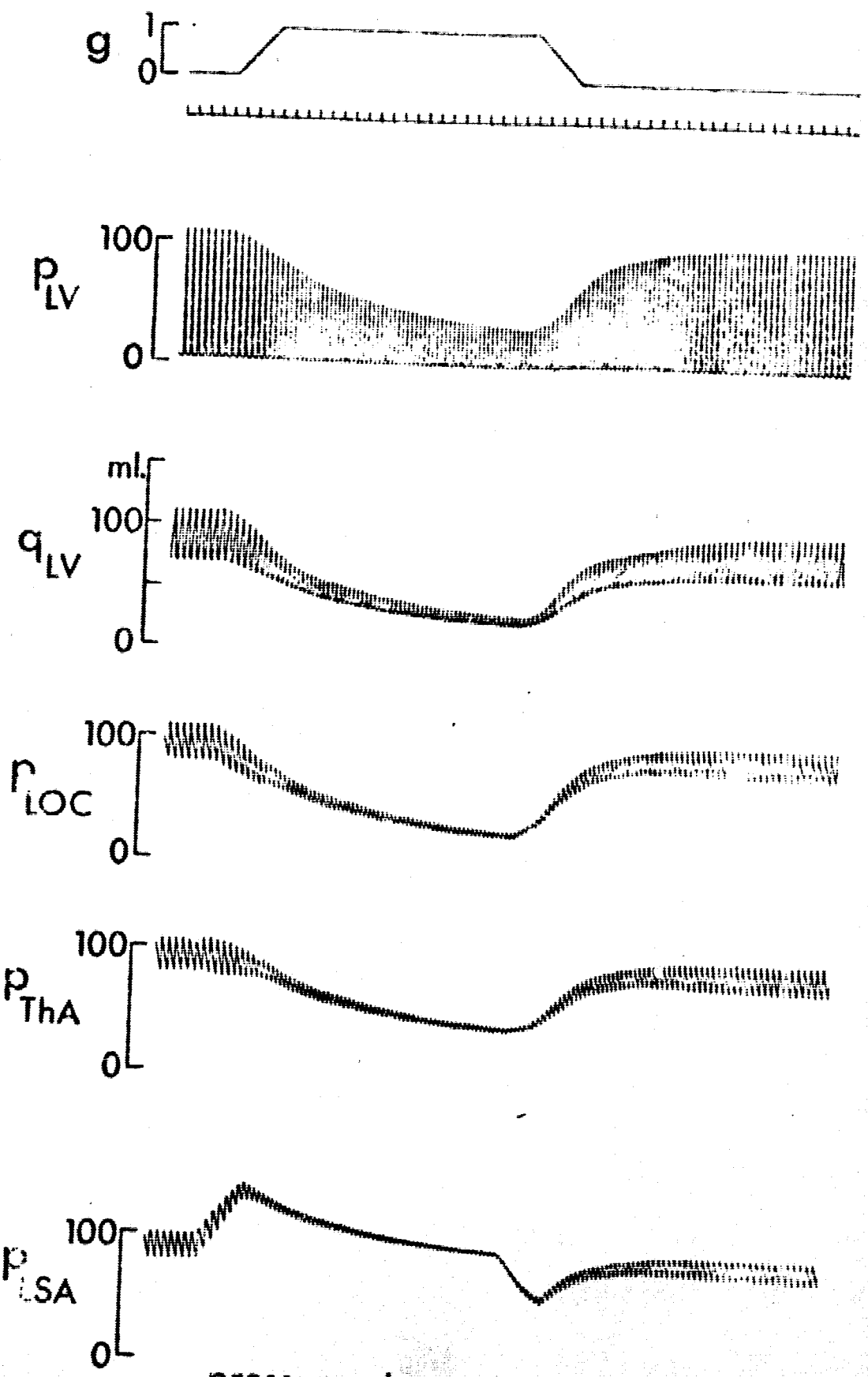
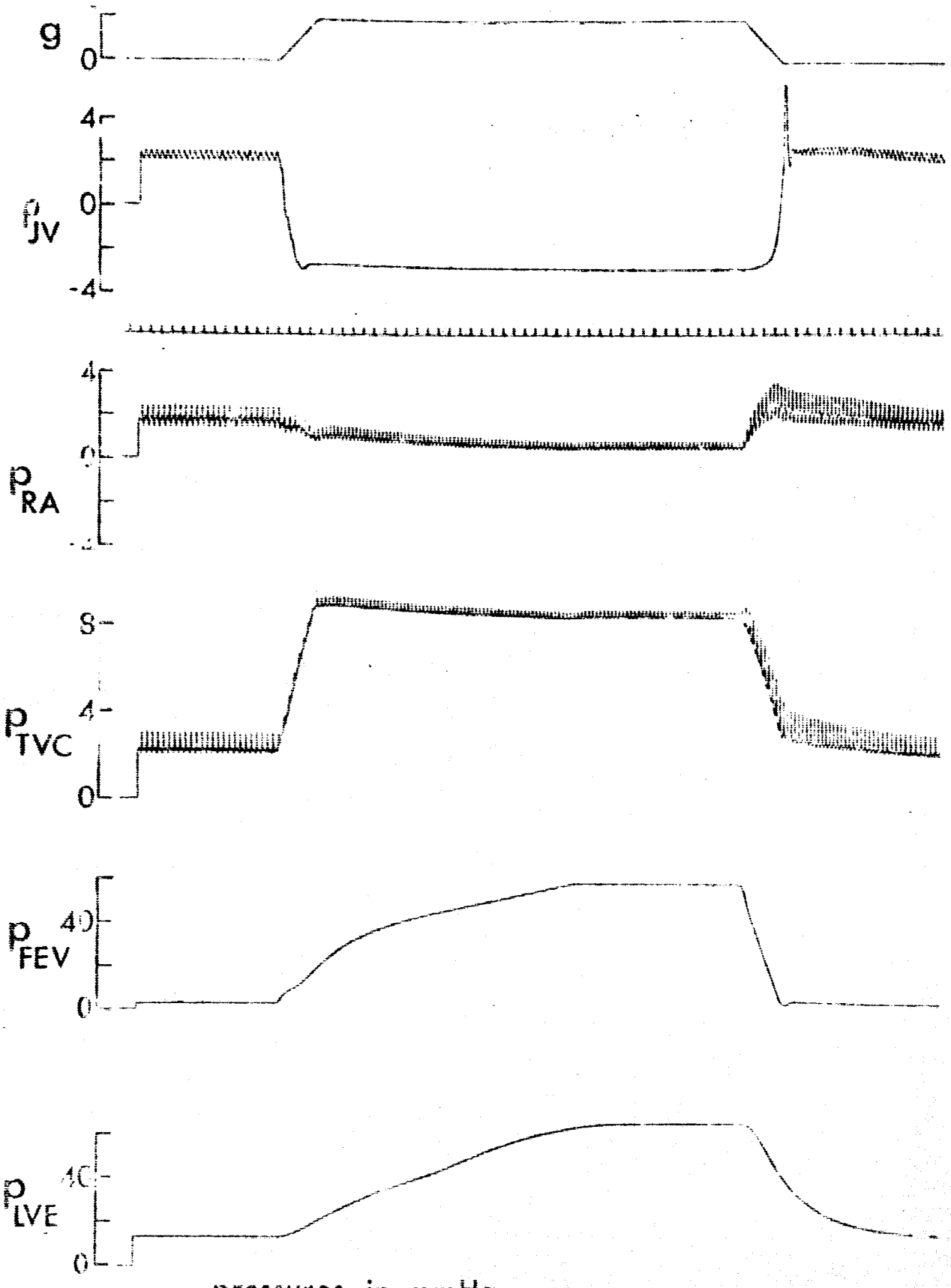
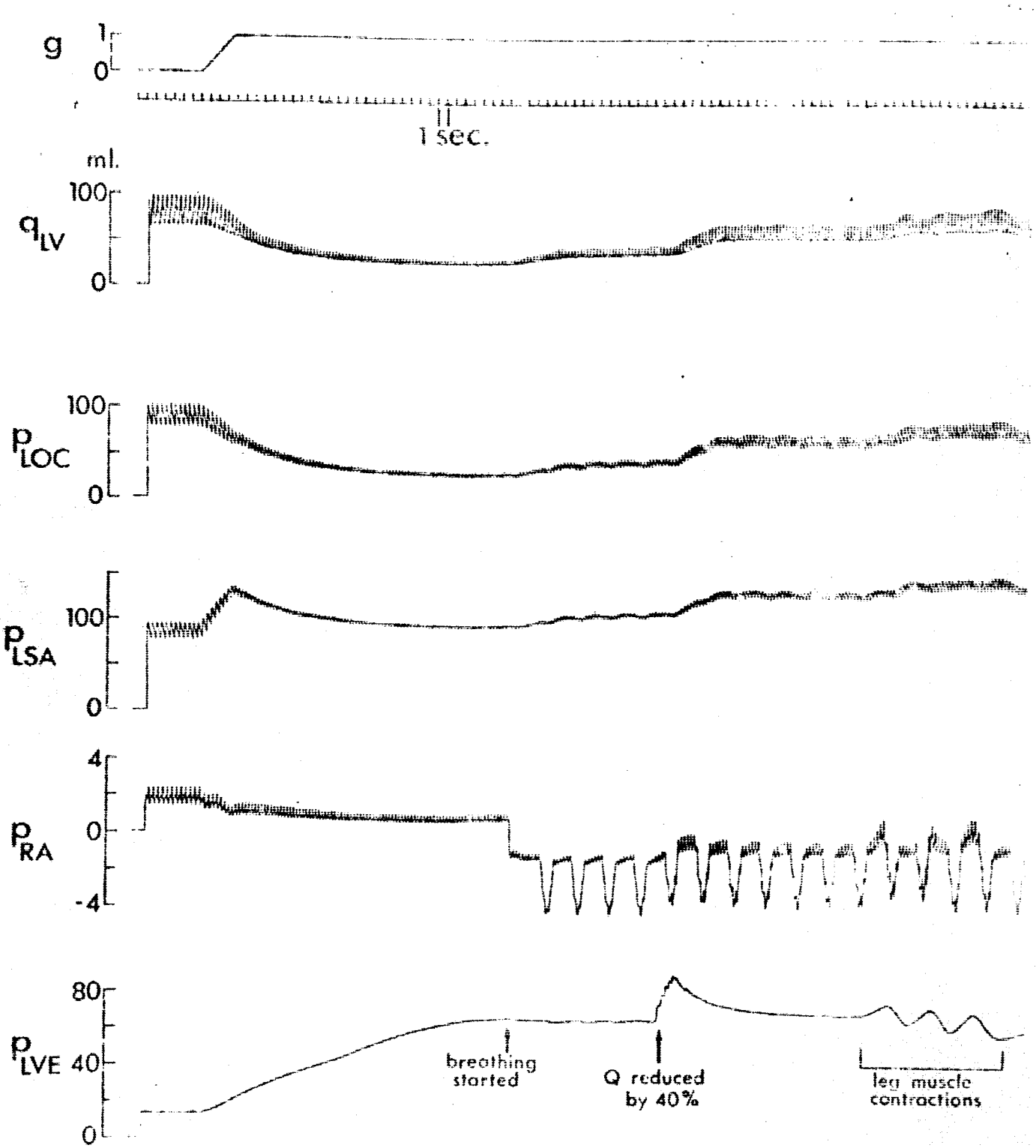
0
-2
-4
-6
time
3.33 sec.
breathing rate = 18/min



(a) pressures in mmhg (b)
volumes in ml.


g
1
0
p_{LV}
100
0
ml.
q_{LV}
100
0
r_{LOC}
100
0
p_{ThA}
100
0
p_{LSA}
100
0
pressures in mmHg


g
0
4
0
-4
p_{JV}
4
0
p_{RA}
8
4
0
p_{TVC}
40
0
p_{FEV}
40
0
p_{LVE}
pressures in mmHg



PRESSURES IN mmHg